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3 - JAN 1959
MADRAS

THE MADRAS CLINICAL JOURNAL

JOURNAL OF THE MADRAS STATE BRANCH INDIAN MEDICAL ASSOCIATION
(With which is incorporated the "Miscellany")

Vol. XXV DECEMBER 1958 No. 6

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Vol. XXV] DECEMBER 1958 [No. 6

REVIEW OF DERMATOLOGY (*Continued*)

by

Dr. T. V. VENKATESAN, M.B., B.S., F.C.C.P., F.D.S. (London) F.A.I.M.

Bacterial Infection of the Skin.

I. Impetigo :

(*a*) Impetigo cantagiosa and its annualar and circinate variants.

(*b*) Impetigo bullosa and its extensive variant pemphigus neonatorum.

II. Folliculitis

(1) Superficial (*a*) Bockhart's folliculitis. (*b*) Acne necrotica miliaris.

(2) Deep folliculitis : (*a*) Sycosis vulgaris. (*b*) Pyoderma faciale. (*c*) Folliculitis decalvans. (*d*) Perforating folliculitis of nose. (*e*) Acne varioliformis. (*f*) Hordeoleum. (*g*) Furuncles and carbuncles.

III. Ecthyma.

IV. Paronychia.

V. Erysippeloid.

VI. Infections of the apocrine sweat apparatus.

I. Impetigo Contagiosa.

Aetology: Micrococci and betahaemotytic streptococci

Age: Common in Children.

Predisposing factors: Anaemia, malnutrition and poor hygiene.

Skin lesions: Appear as small reddish macules which soon become vesicles. These rupture; secondary infection and crust formation take place, The crust is the hall mark of the disease. The commonest sites are face around the mouth and nose, hands and other exposed areas. The lesions spread by autoinoculation. Hence we can see daughter and grand daughter lesions. If the crust is not removed promptly the lesions may spread beneath the stratum corneum.

Treatment: 1. Remove crusts with 2% soda sulphate or 2% soda bicarb.

2. Application of 2% watery solution. Gentian violet or any antibiotic cream. The antibiotic should not be used more than 7 days to prevent the risk of allergic hypertensitiveness.

3. Systemic antibiotic therapy is also advised.

Bullous impetigo is more common in infants and is due to coagulase positive staphylococci. The bullae burst with secondary infection and crust formation. The treatment is similar to impetigo contagiosa.

II. Follicullitis

This is pyoderma originating in the hair follicle.

1. (*a*) Bockhart's follicullitis.

The commonest site is between knees and ankles.

The measures are :

External :—Brilliant green ½%
Mercury perchloride ½%
Spirit 99%
Mix

Injection: Longacting penicillin
Benzathine penicillin 6 lac.
I. M. every third day for as much as 72 lac.

(*b*) Acne necrotica miliaris is a chronic recurrent form of follicullitis of the scalp and improves with treatment similar to Bockhart's.

2. Deep Follicullitis.

(*a*) Sycosis barbae is a purulent follicullitis of the beard and moustache area due to staphylococcal infection.

The measures are neomycin or aureomycin ointment external after warm boric compress. Vioform cream is also good. Avoid shaving and after clipping hairs close with clippers the cream has to be applied. A course of parenteral antibiotic therapy is also necessary.

(*b*) Pyodermia faciale occurs due to staphylococci in young women producing erythema, cysts and numerous abscesses on the face. The absence of comedone, and acneform lesions distinguishes the same from Acne Valgaris. The measures are systemic and parenteral antibiotic therapy.

(*c*) Follicullitic decalvans or Quinquad's disease in the occurrence of minute abscesses and pustules in the scalp finally leading to scar leaving alopecia. The disease is due to staphylococcous aureus. The disease is recalcitrant to treatment. Bacitracin topically is helpful. Massive antibiotic therapy is essential. Before applying bacitracin shampoo scalp with Hexachlorphene.

(*d*) Perforating follicullitis of the nose is an extremely uncommon condition of the vibrissae of the external limits of the nares and responds to antibiotic therapy.

(*e*) Acne varioliformis is due to staphylococcus aureus producing small red swellings around the orifices of the hair follicles which are soon surmounted by pustules. The cases respond to aureomycin ointment and sulphathiazole orally. The character of the pustules and scarring suggest varioliform.

(*f*) Hardeolum is stye occurring in the hair of the lid margin.

(*g*) Furunculosis are crops of boils. The predisposing factors are diabetes, debility and focal sepsis. Staphylococcus pyogenes aureus is the causative agent.

The measures are (1) Treatment of the predisposing factors (2) a course of antibiotics. Erythromycin is the best.

Ecthyma.

This begins in the same way as impetigo. The process erodes the epidermis and produces a shallow ulcer. Betahaemolytic strepticcocci initiate the disease. The lower limb especially the legs are more commonly affected. Topical and parenteral antibiotic therapy are the measures of choice.

Hidradenitis supparative is abscess in relation to apocrine sweat glands. Both strepto and staphylococci are responsible

The lesions occur as small subcutaneous nodules which develop into abscess. The usual site is the axilla. The moist site favours auto infection. Incission and drainage and X-ray therapy are useful.

Dermatological Viral Diseases.

1. Herpes simplex.
2. Herpeszoster.
3. Kaposi's varicelliferimis.
4. Molluscum contagiosum.
5. Warts.
6. Orf.

Herpes Simplex: is the most ubiquitious of all viral diseases. The virus of Herpes simplex produces various manifestations.

(*a*) Skin:
   1. Herpes Simplex.
   2. Eczema Herpeticum.
   3. Traumatic Herpes.

(*b*) Muco cutaneous junction.
   1, Herpes labialis.
   2. Herpes progenitalis.
   3. Valvo vaginitis.

(*c*) Mucous membrane.
   1. Acute gingivostomatitis.
   2. Recurrent stomatitis.

(*d*) Central nervous system.
   Meningo encephalitis.

(*e*) Conjuctivitis and kerato conjunctivitis.

(*f*) Generalised infection.
The virus produces multilocular vesicles deep in epidermis and in the edge of the vesicles ballooning degeneration of the epidermal cells can be seen. The intranuclear inclusion bodies are known as Lipschutiz bodies.

Treatment: 1. Broad spectrum antibiotics.

Internally: 2. Zephiran (1 in 1000) externally.

Herpeszoster: or Singles: Hyperisthesia- pain and itching occur followed by the occurrence of groups of vesicles. Two types occur namely primary and a secondary type following trauma. Two types of degeneration occur in Herpeszoster namely ballooning degeneration and reticular degeneration.

The special types of Herpeszoster are in relation to.

1. Opthalimic division of the 5th nerve.

2. Facial nerve involvement due to geniculate Herpes. (Ramsay Hunt's syndrome).

Complications of Herpeszoster are:

1. Postherpetic neuralgia.
2. Paralysis temporary or permanent.
3. Secondary infection.
4. Ocular involvement due to the 5th nerve first division involvement.

Varicella and Herpes' Zoster are different manifestations of the same virus.

Treatment in Herpeszoster: The following measures have been found useful.

External:—2% methylene blue in alcohol.

Internal:—1. Prednisolone tab. 30 (each 5 mgm.) in gradually decreasing doses.

2. Tetracycline capsules orally one capsule thrice daily for 7 days.

Injections For postherpetic Neuralgia:

1. Soda salicylas injections.
2. Vitamin B12 in massive doses.

Kaposi's Varicelli Form Dermatitis is a disseminated cutaneous herpes simplex. The condition resembles eczema vaccinatum but the inclusion bodies are nuclear and not cytoplasmic. The condition responds to broad spectrum antibiotics.

Molluscum contagiosum is a viral infection of the skin and less commonly conjunctiva. Small sessile peduneulated, umbilicated tumours usually multiple occur. The disease is contagious, several members of one family may be affected. The histopathological specimen shows that the lobules are pear shaped with the apex upwards. The lobules contain masses of ovoid cells derived from prickle cell layer by a special transformation under stimulus of a virus. In the cytoplasm of these cells vacuoles appear containing central masses of inclusion material rich in desoxvribonucleic acid with numerous minute granules. These granules are the inclusion bodies.

Treatment: The growth is to be destroyed by diathermy or incised and the contents squeezed out. Haemorrhage is stopped by pressure, or by swabbing with 3% trichloracetic acid. When the lesions are numerous a course of broad spectrum antibiotics is helpful.

Warts or Verrucae.

1. The Common wart is known as verruca vulgaris and is seen on the fingers and hands. It varies in size from a pin's head to a pea. The colour is yellowish brown or browish black.

2. Verrucafiliformis is thread like pointed excrescence pale pink in colour.

3. Verruca digitata is a wart in which numerous finger like processes arise from an area of the size of a pea. The base is often constricted.

4. Juvenile plane warts are yellowish in colour occurring commonly on the face and dorsum of hands.

Verruca plantaris are flat warty growths of brownish colour and are often multiple and are most commonly seen on the ball of the foot and heal. The lesions are tender. The pain is worse in the early part of the day. They are mistaken for corns. When the warts are cut minute bleeding points can be seen. These are haemorrhages from the capillaries in the elongated paillæ.

Treatment: External:

1. R/-Glacial acetic acid 10 parts
   Strong tincture of iodine 10 parts
   Salicylic acid 20 parts
   Spirit methylated 100 parts
   Add

2. X-ray therapy.

3. Paint the areas with liquor potash and give magsulph ionisation 1%

4. 3% formalin is advised for planter warts for three weeks.

Injections: Bismuth subsalicylate 1 cc I. M. once weekly for 8 weeks.

Orf: This is a contagious pustular dermatitis occurring in sheep rearing areas with a self limited course.

Parasitic Diseases.

Scabies is caused by the itchmite. The mite has four pairs of legs, two pairs anterior ending in suckers and the two pairs behind ending in spines. The impregnated female forms a burrow in the horny layer and lives for about two months and lays about two to three eggs per day. The eruptions are polymorphous producing follicles, urticarial papules, vesicles, excoriations and pustule due to secondary infection. The lesions are commonly seen in the webs of the fingers, ulnar border of hands, anterior axillary folds, buttocks and public region.

In Norvegian scabies myriads of scabeitic lesions occur enveloping the whole body. This is seen in mental patients, geriatric patients and in Hansen cases,

Treatment: This depends on if the skin lesions are infected or non infected. Every member of the family should be treated. A preliminary bath with warm water and soap and scrubbing with rubber brush to lay open burrows.

The following external applications can be used.

(*a*) Unguentum sulphuris.

(*b*) Danish ointment.

(*c*) Benzyl Benzoate emulsion 25%.

(*d*) Tetmosol lotion.

(*e*) Eurax.

During the infected stage secondary infection has to be treated with antibiotics before using above application.

Pediculosis capitis: The infestation with louse produce itching and secondary infection. The treatment is clipping hairs close or shampooing hairs and applying 5% D. D. T. preparation as Benzoped or uniscaboint. Pediculus corporis is important in the dissemination of typhus and relapsing fever and the treatment is similar to above.

Prediculas pubis is shorter and stouter and produces itching and excorcation and treatment is similar.

Disorders of Skin Appendages:

Sweat glands.

Sudamina: Due to fever there is excessive sweating and this produces obstruction of the sweat glands and producing rashes similar to grains of sago. This requires no treatment. The horny layer exfoliates and the fluid is discharged.

Hyperidrosis: or excessive sweating may be generalised or localised. Emotional hyperidrosis is the most commonest form and involves palms and soles and responds to tranquilisers and anticholinergic drugs as Banthine and Pranatal. Banthine bromides is given in doses of 50 mgm. thrice daily.

Localised Chromidrosis: Coloured sweat is produced in this condition. The quantity is minute. The coloured sweat is derived from apocrine glands in the axillae. The part should be kept clean and sponged with 1 in 1000 perchloride of mercury.

Disorders of Hair Follicles.

Hypertrichosis is growth of hair in abnormal situations. In such cases we have to rule out endocrinal causes as adrenal cortical tumours.

In others, the measures are diathermy coagulation or barium sulphide solution locally as depilatory.

Various measures: of beautifying hair and keeping it in a healthy condition have preoccupied man kind since antiquity. Women especially like to have curly hairs. The preparations containing thioglycocollic acid are helpful.

Gray hairs: are due to inability to liberate melanin. The process of inhibition may be temporary or permanent. The treatment is really a problem but various dyes have been tried. The administration of large doses of pantothenic acid and para aninobenzoic acid is helpful.

Alopecia or Baldness may be due to scar formation or noncicatrical as in ringworm infection due to fevers. drugs as folic acid antagonists and aging.

Alopecia areata is a special entity with round or oval patches. This may be seen in the scalp, moustache or beard area eyebrows, axillae and pabic area. The onset is abrupt. There is no inflammation accompanying the loss of hair. A coincident sign in many cases is a variable dystrophy of nails.

In the etiology of alopecial areata profound psychological conflict plays an important part.

Treatment: Stimulating application as follows externally:

R/-	Acid Lactic	dr.	6
	Oleum Recni	dr.	2
	Spirit Myrciae	dr.	6

MIX

Ultraviolet exposures erythema doses are helpful.

Tincture cantharidis is derived from cantharidis vesicatoria or blistering beetle is a helpful local application.

Corticosteroids will temporarily cause regeneration of hair but the effect is short lived.

Disease of Seborrhoeic Glands.

Acne Vulgaris is a classical stigmata of adolescence. The increase in size of sebacious gland and the concomitant increased secretion of sebum during adolescence are physiologic. Over this, inflammatory change occurs. There is obstruction to outflow of sebum producing a blockhead or comedo. Papules and superficial pustules may occur. The bacilli responsible are Acne bacillus and staphylococcus.

The course of Acne: As adolescence passes most acne fades. This is called tempos varos curat or time cures acne. The factors that influence acne are:

(1) Improper diet.

(2) Excorcation.

(3) Fatigue and nervous exhaustion.

(4) Hormonal influence as during menstrual periods.

(5) Warm humid climates.

(6) Bacterial infection.

Treatment of acne: The following mneumonic is helpful.

(A) Correct Anaemia and also give Vitamin A.

(C) Correct Constipation and also give Vitamin C.

(N) Never use creams and snows. Use keratolytic as

R/-	Sulphur	gr.	20
	Salicylic acid	gr.	20
	Parafinum molle	oz.	2

Eskamel is a useful preparation; if there is much inflammation Neomycin with Hydrocortisone as Neocortef is helpful.

(E) Exercise to improve health.

Endocrines: Stilboesterol 0·5 mgm daily for 10 days after cessation of menstrual flow. Three such courses are given. For men stilboesterol 0·25 mgm daily for 6 weeks.

Chocolates and sweets are culprits that aggravate acne and must be stopped.

Disorders of pigmentation.

Vitiligo or Leucoderma: In this condition there is failure of the skin to form melanin. The etiology of this is unsettled.

Treatment advised: – Internal – From Ammi majus is derived the substance Ammoidin and ammidin. These are available as meladinine.

Dose: One tab thrice daily. Each tablet contains 7·5 mgm of ammodin and 2·5 mgm of ammidin.

Externally apply meladinine solution and give UVR exposure.

Melanoderma: The excessive production of melanin produces melanoderma. Increase of melanin occurs in chronic skin diseases and in Addison's disease. The production of melanin is under the control of M. S. H. of the anterior pituitary and in Addison's disease the excessive pigmentation is due to M. S. H. Localised melanoderma is treated with 5% monobenzyl ether of hydroquinone externally and ascorbic acid internally.

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Psoriasis: This is a papulosquamous condition. The disease is of unknown etrology. The following theories are:

(*a*) Toxic.

(*b*) Metabolic: In psoriatics there is increase in blood cholesterol.

(*c*) Infective: Dr. Krylebelieves in a filter passing virus though others do not agree.

(*d*) Psychosomatic: Psoriasis is influenced by the stock market.

(*e*) Endocrinal: Psoriatics get worse during puberty and menopause due to endocrinal upset.

(*f*) Trauma: In posriatics, freshlesions develop at the site of trauma. This is called koebner's phenomenon.

The site of lesions: Scalp, back of elbows, front of knees, small of back are the commonest sites.

On scraping, the silvery scales are found. At the end of the scales a membrane with petechial haemorrhages are found. This is called Buckley's membrane.

Types of psoriasis: According to the type of lesions, various types are described:

1. Psoriasis punctata: Small punctate lesions.
2. Psoriasis figurata: Like figure.
3. Psoriasis Arthropathica: Psoriatic lesions with joint pains.
4. Psoriasis nummularis: Coin like lesions.
5. Psoriasis annularis: Like annular lesions.

Treatment:

1. Control psychosis by tranquilizers like Benactyzine 1 mgm thrice daily.

   Meprobamate 400 mgm or Chlorpromazine 100 mgm.
2. Reduce fat in diet.
3. Corticosteroids: Prednisolone in gradually decreasing doses starting with 20 mgm first day is helpful especially in psoriasis arthropathica.
4. Externally:

R/ –	Liquor picis carb	dr. 1
	Hyd amm.	gr. 20
	Salicylic acid	gr. 40
	Paraffin ointment B.P.	oz. 1

Mix and make into ointment.

5. UVR Exposure.

Lichen Planus: This is characterised by papular lilac coloured eruptions, and polygonal in shape. If the surface is moistened with oil and water, white opalescent striae forming a fine network are visible. These are called Wickam's striae. The eruptions occur on the face, scalp, flexor surface of forearms, wrists, the front of the legs, innerside of the thighs and hips. The mucous membrane of mouth may be involved. Itching as a predominant feature.

Aetiology: Barber believes in a virus etiology. The rapid response to ACTH is in favour of allergic etiology. The disease commonly occurs between 30 and 40 years and women are more affected than men. The measures of treatment are:

1. Antipruritic lotion externally.
2. Corticosteroids.
3. Large doses of Aneurin
4. PAM has been found useful in a number of cases.

Linchen Nitidus. Pin head sized papules occur on the genitalia, abdomen and breast. There is slight itching and lilac colour of Lichen planus is absent. The disease is unresponsive to treatment.

Tubercular Diseases of Skin: The following classification is helpful.

(*a*) True cutaneous tuberculosis (Lesions contain tuberclebacilli)

(*b*) Tuberculids (Allergic origin: No tubercle bacilli in lesions).

A. **True Cutaneous tuberculosis :**

1. Primary: (No previous infection. Individuals tuberculin negative in initial stages).

(*a*) Primary inoculation Tuberculosis. (Exogenus implantation of tuberculosis into skin)

(*b*) Miliary tuberculosis of the skin (Haematogenous dispersion).

2. Secondary tuberculosis (Lesions develop in person already sensito tuberculin).

(*a*) A result of prior lesions. Tubercle bacilli difficult or impossible to demonstrate).

(*a*) Lupus vulgaris : Inoculation into skin from external or internal sources.

(*b*) Tuberculosis Verrucosa cutis - same as above.

(*c*) Scrofulodermia : Extension from underlying focus inbones or glands.

(*d*) Tuberculosis cutis orificialis; Mucous membrane lesions.

Tuberculids :

1. (*a*) Lupus miliarisdisseminated facei

(*b*) Papulonecrotic tuberculids.

(*c*) Lichen scrofulosorum : Follicular papules or Lichenoid papules.

2. Granulomatous : Erythemaindu-ratum or Bazin's disease.

Primary Tuberculosis of Skin.

1. **Primary inoculation tuberculosis:** The lesions commence as a nodule which ulcerates and finally produces a tuberculosis chancre which has nondescript appearance. The histological picture is acute inflammatory reaction with numerous tubercle bacilli.

2. **Miliary tuberculosis of skin**: The lesions are macules, papules, visicles and purpuric spots. Organism are plentiful.

Reinfection tuberculosis : Lupus Vulgaris : The disease occurs in children and young people commonly occurring on the face and cheek and less commonly on the extremities and trunk. Traditionally the nodules are yellowish brown. It has a translucency and looks like apple jelly when pressed with a slide.

The nodules coalesce to form plaque like lesion. During a course of years large serpiginous and gyrate plaques form. In mucous membranes the lesions are vegetative or papillary growths which ulcerate and produce irregular scars.

Histopathology: Typical tubercles with epithelied cells, giant cells and peripheral zone of lymphocytes. Caseation necrosis is slight. Tubercle bacilli are present in small numbers.

Tuberculosis Verrucasa Cutis. Warty horny lesions occur. There is peripheral spread and central scarring. Histological picture :

1. Acanthosis ie. increase in thickness of stratum Malphigi.

2. Hyperkeratosis ie. thickness of horny layer.

3. Papillamatosisie upward proliferation of papillæ.

4. Beneath epidermis acute inflammatory exadate of polymorphs and lymphocytes.

5. Tubercle bacilli are more numerous than in lupus vulgaris.

6. Cessation is more than in lupus vulgaris.

Scrofuloderma : Extension of tubercular process to skin from below as gland. The skin breaks and ultimately undermined ulcer are formed.

Histopathology: The centre of the lesion shows nonspecific abscess formation with ulceration of epidermis. At the periphery tuberculoid structures can be seen.

Tuberculosis cutis orificialis : The lesions develop around the mucocutoneous junctions. The sites of prediliction ioral and genital areas. The ulcers are ndolent, round and shallow.

Histological picture is chronic inflammatory tissue and tubercle formation.

Tuberculids : These lesions represent reactions of hypersensitivity and tubercle baccilli are always absent. The following criteria are prerequisites to diagnosis of tuberculid.

1. Positive tuberculin test.
2. Tuberculoid histological structure.
3. Active tubercular focus.

A. **Papular forms** : 1. Lupus miliaris disseminated facei :

The eruptions are crops of well defined soft papules. The papules are discrete Finally pitted scars are left following involution.

The histological picture shows typical tubercles with central cessation. Lichenoid tuberculid is only a variant of the above.

2. Rosacea like tuberculid of Levandowsky : The lesions are found on the peripheral third of face. The papules show islands of epithelial cells with few lymphocytes and no giant cells.

3. Papuloneerotic tuberculiods : There are deep seated papules typically capped by a pustule. On the face it is known as acnitis ; on the hands known as folliclis.

The Histological picture shows small central area of neerosis surrounded by non specific inflammation. The blood vessels in the lower dermis shows obliterative endarteritis.

4. Lichen scrofulosorum : Commonly occurs in children. Mostly seen with tuberculosis of the glands, bones and joints. The lesions are acuminate papules with brownish tint.

The infiltrate contains epitheloid cells with occasional giant cells. A Zone of lymphocytes is seen at the periphery.

B. **Granulomatousform** : This is a chronic recurrent eruption occurring in the calves of women. The lesions are painless deep seated nodules and gradually extend to the surface and ulcerate.

Histopathalogy : The infiltrate is limited to the lower dermis and subcutaneous area. Both types of infiltrate may be present namely specific and non specific. They infiltrate between fat cells and gradually replace them. This is called Wucheratrophic of fat. The blood vessels show proliferative changes and thrombosis.

Dietetic treatment in cutaneous tuberculosis : The Principles are :

1. Lack of salt.

2. Inclusion of large amount of fresh vegetables and fruits and alteration in the percentage of fats, proteins and carbohydrates. On this is based Gerson's diet and Hermonsdorsfer's diet.

The salt restriction produces a relative cutaneous dehydration thus forcing salt depot to give their supply with the consequence of the exudative processes regressing.

Dr. Koertz believes that salt restriction produces a redistribution of minerals and increase of calcium.

The various measures of treatment are:

1. Local therapy:
   - (a) UVR Therapy.
   - (b) Electro surgery.
   - (c) The acid nitrate of mercury externally.
   - (d) Intralesional streptomycin.
2. Systemic therapy:
   - (a) Vitamin A and D. Prof. Dowling used Charpy's method of using massive doses of calciferol or Vitamin D 2 15,000 I. U. daily.
3. **Chemotherapy in Tuberculosis:**

1. I. N. H. is given in doses of 150 mgm daily for three months.

2. Streptomycin
   - (a) 1 gm. thrice weekly I M.
   - (b) Local infiltration of streptomycin 0·5 gm into the lesions.

3. Combined streptomycin and I.N.H.

Method: 1 gm. I. M. of streptomycin thrice weekly and 150 mgm of I. N. H. orally daily.

4. P. A. S. may be used in combination with I. N. H. or streptomycin

Pemphigus: Pemphigus is an essentially bullous skin lesion. Great advance in knowledge is aided by histopathological studies of the lesion. The varieties of pemphigus are:

1. Pemphigus Acutus.
2. Pemphigus Vulgaris.
3. Pemphigus foliaceus.
4. Pemphigus Vegetans.
5. Pemphigus Erythematoides.
6. Familial Benign chronic pemphigus of Hailey and Hailey.

1. **Pemphigus Acute:** In this there is severe constitutional symptoms with bullae and cortisone or A. C. T. H. is benificial.

2. **Pemphigus Vulgaris:** Involves the skin and mucus membrane. The bullae arise on normal skin with a zone of eryethema around the bullae. The bullae contain acantholytic cells. These cells show degenerative changes and are known as Tzanck cells.

3. **Pemphigus foliaceus:** Is a rare type with the bullae rupturing and producing on exfoliative lesion.

4. **Pemphigus Vegetans:** Contain vegetative plaques and a few bullae. These plaques are made of micioabscesses.

5. **Pemphigus Erythematosus:** Is known as Senior-usher syndrome and is a benign type of chronic Pemphigus resembling Lupus Erythematosus.

6. **The Familial Benign chronic Pemphigus of Hailey and Hailey:** Occurs as vesicular and bullous lesions and are distinctly familial in origin.

Tzanck cells are not found.

Treatment of Pemphigus: The measures are Hydrocortisone ointment locally and Hydrocortisone lotion locally (EFCORLIN) and systemic administration of prednisolone and prednisone.

In this article Dermatology has been reviewed, not in toto, but to a large extent. This subject throws light on the necessity for research in the field. It is, therefore, essential to keep abreast with the advances in this subject.

(Concluded)

* DISSEMINATED SCLEROSIS.

by

DR. G. VICTOR, M. D.,

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Definition : Disseminated Sclerosis is a disease of unknown aetiology,• with patchy demyelination followed by gliosis as essential pathology, characterised by early symptoms in the form of focal lesions of the nervous system, & late symptoms in the form of nystagmus, dysarthria, intention tremor, & ataxic paraplegia and classical remissions and relapses.

Introduction : This disease is also called Multiple Sclerosis, Insular Sclerosis. It was first described in 1835. Next to Syphilis, this is the most common organic disease of the nervous system in Europe. All attempts to reproduce the disease have failed.

Aetiology : (a) *Geographical distribution* : The significance of the geographical distribution is important. There is an uneven distribution of Multiple Sclerosis throughout the world. The malady is found in persons of widely varying occupation and in all strata of society. The likely explanation may be (1) racial susceptibility to the disease, low prevalence in equatorial and sub-tropical regions The evidence that race determines the geographical distribution of Multiple Sclerosis is inconclusive. (2) Diatery factors such as average daily intake of fat exceeding 100 gms. (3) Deficiency in the diet. (4) Abnormal mineral content of the soil and therefore of the food, e.g., lead (workers found a higher lead content in the teeth).

(*b*) *Family History* : Occasionally Disseminated Sclerosis may be met with as occurring in more than one member of a family. Gowers said that " direct heredity or the affection of two brothers or sisters has been noticed in a few instances, but it is quite exceptional ". McAlpine observed a familial prevalence and feels that an inherent constitutional factor might render a person more susceptible to the illness. According to Mackay, familial tendency is observed in Disseminated Sclerosis, which is due to a familial constitutional vulnerability to the disease. Pratt, Compston, and McAlpine concluded that a genetic factor is present in Disseminated Sclerosis allowing the causitive factors to act more readily. Pratt is of opinion that " the fact that Disseminated Sclerosis does not always appear in both members of an identical twin pair and the frequent difference in age of onset when both are affected. suggest that exogenous factors play an important part in the development of the disease ". There has been a rare example of the disease occurring in three generations. Conjugal Multiple Sclerosis has been reported.

(c) *Age of onset*: It is observed ihat the disease has occurred in the 10th year of life in the youngest patient. When symptoms commence after 50 particularly when they are progressive, diagnosis must be carefully made.

(d) *Sex of incidence*: Twenty of the reports showed a predominence of males and six (including that of Charcot) a preponderence of females.

(e) *Factors in the past Medical History*:

1. Infection: The cause is an infection to which certain people are pre-disposed. Papez recently demonstrated " Inclusion bodies " in the affected

* Lecture delivered at the Indian Medical Association, Madurai.

neurones, this suggests the concept of Infection. According to Nielson, it is an infection with an allergic response to that infection. Superfical infection in the form of whitlows, boils, carbuncles, infected cuts, bites, and burns was met with in Disseminated Sclerosis patients, prior to onset of the illness. Steiner has reported that infection by a spirochete could cause Disseminated Sclerosis.

2. **Allergy** : Under this heading the following conditions are suggested to be capable of producing this illness, viz., Asthma, Hay fever, Urticaria, Angio-neurotic Oedoma, Food Allergy, Contact Dermatitis and Eczema.

3. In about 20% of McAlpine's cases, *lowered state of general health* was the chief observation before the illness began. Fatigue and physical exertion often precipitate symptoms.

4. Signs of illness may appear after a severe *trauma*, especially direct injury to the skull or spine. The following was the recommendation made by the Association for Research in Nervous and Mental Diseases in 1922 :— " In a small percentage of Multiple Sclerosis it appears to be excited by trauma, but trauma cannot itself cause it, but may apparently awaken a disease process already potentially existent "- The time interval between trauma and onset of symptoms was not over two months. There are also instances of a peculiar relationship between the site of injury and the location of the initial lesion. Thus it could be concluded that trauma (including surgical operations) could precipitate Multiple Sclerosis in a predisposed person or cause a relapse.

5. **Pregnancy** : May either precipitate the disease or influence its course. Occurrence of Multiple Sclerosis and pregnancy together is highest during the second decade.

6. It was also observed that Blood Transfusions, Inoculations, and Toxic Drugs such as Arsenic, could precipitate symptoms in those who are already suffering from the disease.

7. **Anxiety and emotional disturbance** play a prominent roll in the onset or relapse of Multiple Sclerosis.

8. **Chill or Low temperature** could occasionally precipitate the condition.

9. **Precipitating factors** : Influenza, Upper Respiratory Infection, Specific Fevers, Sepsis, Puerperium and lactation, Dental extraction. Carbon-monoxide poisoning, Electric shock could produce preciptation of the disease.

(f) Maiqulis, Soloviev, Shubladze have suggested *Filterable Virus* as a causative agent.

(g) The modern view is that the glial overgrowth is either a scar reaction to the previous inflammatory and demyelinating process or it is caused by the same agent which destroys the myelin sheaths.

(h) Multiple Thrombotic lesions of unknown cause.

Pathology : The condition starts as a circumscribed patch of nervous tissue showing at the beginning, destruction of the myelin sheaths of the nerve fibres and to a much less extent of the axis cylinders and ends with the formation of a " sclerotic plaque ". These patches mainly affect the white matter of the brain and spinal cord, sometimes the patches are found in the grey matter of the cerebral cortex, in cranial and spinal nerve roots, and rarely in the grey matter of the spinal cord. The pathological process almost entirely spares the cortex of the cerebrum and cerebellum. Favourite sites for the pathology are :—Optic nerves and chiasma, in the region of cerebral ventricles, sub-pial region of the spinal cord. The plaques of Multiple Sclerosis lie close to the cerebral cortex, but do not affect the

cortex. In the Optic Thallamus, Brainstem, and the Spinal Cord the grey matter is regularly affected exactly as is the white matter. The plaques tend to lie close to the ventricular lining of the brain. In the Pons and Medulla, the plaques tend to come to the surface. Nerve roots, cranial nerves more often as well as spinal nerves, are affected. The Optic nerves are frequently involved. A complete or partial demyelination together with a total loss of Oligodendroglia cells in the plaque is the essential pathology of the condition. Lumsden suggests a breakdown in cellular enzyme systems. A secondary gliosis developes in old foci and some peri-vascular infiltration is found in fresh lesions.

Symptoms and Clinical Signs : The condition presents a varied clinical picture. The onset is usually rapid. In a series of 100 consecutive patients the first complaint was weekness or loss of control over limbs in 50 cases, visual symptoms in 29 cases, sensory symptoms in 11 cases, miscellaneous in 10 cases (to include vertigo, tremor, ptosis, loss of taste, epilepsy, and impotence). Weekness of one or both lower limbs and disturbance of vision are the commonest initial symptoms. Much less often, symptoms appear insidiously. Rarely the onset is fulminating with a picture of Acute Encephalitis, Myelitis, Encephalomyelitis.

(a) *Motor symptoms* may be 1. Loss of power in lower limbs, producing fatigue, heaviness, later, spastic paraplegia. 2. Sudden weekness of one upper limb associated with loss of sense of position in the fingers. 3. Facial weekness and hemiplegia. Inco-ordination is often present - when present in upper limb it produces Intention Tremors (finger – nose test, lifting water to lips) ; when present in the lower limbs it produces ataxic gait ; when present in the head it produces Titubation (in late stages). The intention tremor may be either the medium or the coarse type. Dysarthria due to spastic weekness or ataxia of muscles of articulation, is often noticed. Rarely transitory Aphasia is met with. Tendon reflexes are exaggerated, in the early stages abdominal reflexes are lost in Two Thirds of all cases, & in the late stages 80 - 90% of all cases, show Babinski sign. As a result of involvement of both the pyramidal and the proprioceptive pathways, there is a resulting spastic ataxic gait in Multiple Sclerosis. The ataxia may be both sensory and cerebellar in origin.

(b) *Sensory Symptoms.* Paraesthesiae in the form of numbness and formication are present on one half of the face or a limb. A patch in the Posterior columns of cervical cord will produce a sensation of electric shock radiating through the body on flexing the cervical spine. A plaque in the Column of Burdach in the cervical region will produce asteriognosis of hand. Trigeminal Neuralgia is rare. Objective sensory loss is present in 50% of cases, which is in the form of postural and vibration sensory loss. Occasionally sensory loss with a level on the trunk is seen. There will almost certainly be loss of vibration sensation at the malleoli and probably some impairment of postural sensibility in the toes. '' Multiplex dolorosa' is the term applied to the root pain which may occur in occasional cases of Disseminated Sclerosis. This often misleads to an erroneous diagnosis of spinal tumour.

Ocular Symptoms – Acute unilateral retro-bulbar neuritis, and in 50% of cases the characteristic temporal pallor of the discs is found.

Auditory and Vestibular symptoms – Vertigo occurs as a common and early symptom. Severe vertigo with vomiting and coarse nystagmus may sometime be found.

(c) *Vegetative Symptoms :* Cervical sympathetic palsy, brady cardia or tachycardia, glycosuria, vomiting and if 4th ventricular or 3rd ventricular area is involved, fever will also be present. When the spinal cord is involved, spastic paraplegia characterised by radicular parasthesias, in severe cases involvement of sphincters, ataxia, and loss of abdominal reflexes are noticed. As far as reflexes are concerned, in upper motor neurone lesions, the deep reflexes are increased, in lower motor neurone lesions as well as in acute lesions, they are temporarily abolished. With involvement of meninges, when the head is flexed, an electrical sensation is experienced down the limbs or back. This goes by the term LHERMITTE'S SIGN. The cerebro-spinal fluid shows the following changes. In 50% of cases, there is an increase in the number of lymphocytes during activity. An increase in total proteins or globulin is more constant. In 10% of cases, the mononuclear cells are in excess. The Colloidal gold curve of Lange shows the characteristic paretic (or luetic) curve in 50% to 75% of cases. (The so-called " First Zone Curve "). Bio-chemical changes are rather interesting. 25% of cases show normal figures, 25% show outstanding changes such as pleocytosis, Protein increase, First zone colloidal gold curve ; in 50% of cases slight increase in lymphocytes and a moderate increase in proteins are noticed.

(d) *Other Symptoms* : 1. Bladder - a delay or precipitency of micturition and later, retention or reflex emptying of urine and faeces may occur. Rarely, the first symptom of the disease would be acute retention of urine.

2. Nystagmus is seen in 70% of cases. This is absent on central fixation, but makes its appearance on conjugate deviation.

3. Ocular Palsies - A plaque in the Mid - brain or Pons, will produce paralysis of Conjugate Ocular deviation. With lesion in the Posterior Longitudinal Bundle, dissociation of lateral conjugate deviation occurs. Rarely, paresis of both Internal Recti, Ptosis, Retraction of upper eye-lid may occur.

4. Pupils - Loss of light reflex and preservation of accommodation reflex unilaterally, or total Internal Ophthalmoplegia may be seen. A brain-stem lesion may produce paresis of ocular sympathetic resulting in ptosis, enophthalmos, and myosis.

5. Mental Symptoms include lowered intellect, emotional changes (euphoria, loss of emotional control such as laughing and crying), delutional states, and terminal dementia.

Localisation of lesion and co-related symptoms.

1. Lesion in semi-ovale and frontal lobes will produce disturbances of behaviour, difficult social adjustment, mild or major behaviour disorders, and auditory hallucinations.

2. Affection of Association Tracts will produce asteriognosis, aphasia or apraxia, stammering and stuttering.

3. Affection of Projection system will give rise to Jacksonian convulsions, hemiplegia, asteriognosis, and sphincter disturbances.

4. Lesion in the Thalamus will give rise to emotional disturbance (forced laughing and crying), painful paraesthesias and central pains.

5. Involvement of cerebellar mechanism will result in Charcot's triad in late stages of the illness.

6. Involvement of cranial nerves will produce the following :—

First nerve - Parosmia, Anosmia, Olfactory hallucinations.

Second nerve – Retrobulbar neuritis, loss of vision in one eye with a central scotoma, contracted fields with central or para-central scotoma; with plaque close to the nerve-head, choked disc (rarely), papillo-macular bundle degeneration, amblyopia – unilateral or bilateral; with lesion in Optic chiasma – temporal hemiatrophy (unilateral or bilateral).

Third, Fourth & Sixth nerves–Diplopia, ptosis, anisocoria, sluggish reaction of pupil to light, weekness of lateral gaze or paralysis of upward gaze in cases of Mesencephalic lesions.

Fifth nerve–Facial palsy (Supra – nuclear lesion) or paralysis of the peripheral type.

Eighth nerve – Commonly tinnitus and rarely, deafness.

Ninth, tenth and eleventh nerves–Nausea and vomiting.

Twelfth nerve – Unilateral paralysis of the tongue.

Clinical Types of Disseminated Sclerosis:

(*a*) **The generalized form**: This is common among younger patients. Prominent features are:—pallor of optic discs, nystagmus slight intention tremors, ataxia, weekness with spastic lower limbs and defective spincters.

(*b*) **Spinal forms**: 1. Progressive Spastic Paraplegia (occurring in middle-aged patients) 2. Uni-lateral Spinal lesions-with pathology in the cervical cord. 3. Sacral form - with pathology in Conus medullaris.

(*c*) **Cerebellar; Vestibular; Pontine, and Bulbar forms**: The "brain - stem Multiple Sclerosis" will show diplopia, fifth and seventh nerve palsy, and vertigo.

(*d*) **Acute form**: Fatal in 3 or 4 months fever is present. In severe cases, onset is characterised by head-ache, vomiting, giddiness, and delerium.

Certain text books of Neuralogy give another type of classification. The following is an example:—

1. Cerebral, Cerebellar, and Spinal forms.

2. Acute, Steadily progressive, and Intermittant forms.

3. Wilson's "Subjective Form"—here patients act like a hysteric.

4. McIntyre's Acute, Remittent (which is the Classical Form) and Chronic Progressive forms.

Another type of the disease is described. It is the "Amyotrophic form" of Disseminated Sclerosis, characterised by wasting in forearms, and hands.

Course and Prognosis: In the course of Multiple Sclerosis, remissions and relapses, increasing disability, and premature death are often noticed The interval between relapses (i e., interval between, onset and the first relapse, or between relapses) varies greatly both from case to case and in individual patients. Some patients, especially early in the disease may have four or five episodes in a year and others may have a latent phase of 20 years or more. The Chronic Progressive state occurs in two forms, (*a*) one characterised by progressive downhill from the onset - a mtnority and (*b*) the other, downhill after an initial relapsing pattern of events - a majority.

Working capacity and degree of mobility are the two factors on which the extent of disablement in Multiple Sclerosis is based.

1. **Muller's Classification** (1949):—(*a*) normal, (*b*) slight disablement, (*c*) moderate disablement, (*d*) severe disablement.

2. **McAlpine and Compston's Classification** (1952):–(*a*) unrestricted, (*b*) restricted, (*c*) markedly restricted, (*d*) mobile at home, (*e*) immobile at home and *(f)* bed-ridden.

Spinal puncture may be followed by an increase in clinical symptoms in Multiple Sclerosis. Dr. Furges Furguson, Director of the Neurological Institute of Manchester is in favour of this view.

Direct cause of death – Vital centres in the Central Nervous System are rarely affected causing death, excepting in acute cases who die within a few months of onset of the illness. The common causes of death in a particular series of 57 cases included :— respiratory infection, urinary infection, cachexia, and septic absorption from bed sores, carcinoma, peptic ulcer, cerebral haemorrhage, aplastic anaemia, overdosage of hypnotic, and result of surgical exploration.

The condition may terminate fatally in 3 months or the patient may be able to work for 30 years after onset. Remissions and exacerbations are frequent. 20 years is almost the average expectation of life in fatal cases, however, the end is rather distressing. Thus we see that the prognosis is not always bad. Wilson followed up a case for 30 years and found very little evidence of the disease subsequently. Regarding prognosis as to life, it is worthwhile quoting the words of Byrom Bramwell : " the duration of the disease varies greatly in different cases. The course is slow and chronic, the disease may last for 20 or more years before the fatal termination is reached. In rare cases, the disease persues a rapid course. It is probable that in a few rare and exceptional cases the disease is permanently arrested ".

Diagnosis. This is based on –

1. evidence of more than one lesion in the central nervous system,
2. evidence of progression such as remissions and exacerbations,
3. Characteristic signs of Disseminated Sclerosis such as :— retro-bulbar neuritis, hemiatrophy of optic nerves, combination of motor disturbances and paresthesias, little pleocytosis of the cerebro - spinal fluid, and a paretic gold curve in the absence of syphilis,
4. The triad, Charcot has described as inconstant and late in appearance and is not pathagnomonic for arriving at a diagnosis,
5. On the other hand, appearance of unilateral retrobulbar neuritis in a healthy young adult is more commonly due to Disseminated Sclerosis than to any other cause,
6. A strong positive 1st zone gold curve in the presence of a negative Kahn or W. R. in a patient who was not previously treated for syphilis, is diagnostic of Disseminated Sclerosis.

Differential Diagnosis.

1. Cervical Spondylosis.
2. Dorsal disc degeneration.
3. Spinal cord compression. (tumours)
4. Subacute combined degeneration of the Cord.
5. Motor Neurone Disease.
6. Vascular abnormalities of the brain-stem and spinal cord (Haematomyelia).
7. Familial forms of Spastic Ataxia and Paraplegia (e. g. Friedreich's ataxia, Spino-cerebellar Ataxia).
8. Other demyelinating diseases, viz., Neuromyelitis Optica, Acute Disseminated Encephalomyelitis.
9. Other diseases :—
   (*a*) Meningo-vascular and Parenchymatous Syphilis (Tabes)
   (*b*) Nutritional diseases - Wernicke's Encephalopathy
   (*c*) Pyramido - Cerebellar Degeneration (Subacute and Progressive Cerebellar degeneration)

(*d*) Vascular diseases - aneurysm of middle cerebral artery.

(*e*) Periarteritis Nodosa.

(*f*) Hysteria.

Treatment. There is no specific treatment for Disseminated Sclerosis. These patients ought to avoid fatigue due to exertion and they ought not to overwork. Marriage is to be discouraged and pregnancy avoided. Good deal of encouragement and suggestion is required, and the ultimate prognosis should never be given directly and bluntly to the patient.

Drug Therapy. As a palliative measure, the following may be tried :—

1. Arsenic, intra-venously 0·45 gram weekly for 4 - 6 weeks, to be repeated every few months. Fowler's Solution m. 3, t. d. s.

2. Belladona, gr. ½ as a pill, for bladder weekness.

3. Liver Extract Injections.

4. Dicumarol treatment of Putnam - to deal with Venule Thrombosis.

5. Horton's method - Histamine diphosphate 2·75 mgm. in 250 - 500 c. c. of Normal saline as intra-venous infusion in 2 hours, daily for 15 days.

6. Niacin 50 mgm. 4 times daily by mouth and mixed a, b, y, Tocopherols 50 mgm. 4 times daily. This improves vascularity and enhances recovery of neurones with minimal damage.

7. Glutamic Acid & Phosphovecithin.

Pyrexial Therapy. Protein Shock Therapy (using Malaria, Pyrifer, TAB Vaccine) has been found to be useful.

Other measures.

1. Kabat - Kaiser System consists of keeping the patient on his feet, avoiding exhaustion. However, gentle fatigue is beneficial. If he is permitted to be bed-ridden, he rapidly goes downhill.

2. Massage and passive movements are also helpful.

3. Well balanced & nourishing diet (C. L. diet)

Case Records - Report of 10 Cases.

Case No. 1. Kamalamma, Female, 20 years, complained of heaviness and pain in lower limbs with tremors of body for 6 months and nausea for 3 months. On examination, there was loss of power in right upper and lower limbs, spasticity in all four limbs, inco-ordination was present in left arm, titubation and tremors of fingers were also noticed, tendon reflexes (knee, ankle, triceps, supinator) were exaggerated, plantar response was extensor on both sides, and the gait ataxic. Investigations showed a negative blood VDRL, and normal fundus.

Case No. 2. Ramaswamy, Male, 30 years, chief complaint was inability to walk, of six months duration and in addition he noticed gradual speech disorder, hesitancy of micturition, and visual disturbance for eight months. Clinical examination revealed the following : Speech slurring, diplopia present, visual fields markedly restricted, involvement of VIII nerve on right side, weakness of right leg, increase of tone in both legs, slight inco-ordination of both upper limbs ; sensory loss included : loss of deep touch below knees, of vibration below anterior superior iliac spine, of pain and temperature below knees. Plantars – extensor on right side and irresponsive on right, cremasteric lost on both sides, abdominal reflex lost in all four quadrants, epigastric lost on both sides ; hyperreflexia, plantar clonus (spurious) present on bothsides, gait – spastic, ataxic. Blood Kahn negative, X-ray spine – no appreciable disease, fundus–syphilitic neuro – retinopathic with optic atrophy.

Case No. 3. Ganapathy, Male, 28 years, tremors of all four limbs and weakness of left arm and leg of sudden onset, duration 3 years. Suffered from venereal disease 3 years before present illness. Positive findings were: speech-spastic dysarthria, power diminished in left limbs, tone increased in all four limbs, hyperreflexia present, extensor plantar response on left side, ankle clonus present on both sides (spurious), inco-ordination noticed in all limbs. Investigations revealed a negative serology and slight pallor of both optic discs.

Case No. 4. Ramaswamy, Male, 29 years, complained of tremors of all four limbs with weakness; onset sudden, duration 9 months, symptoms exaggerated during the last five months. Clinically, power was diminished in all four limbs, lead-pipe rigidity was noticed in all the limbs, tremors present in all four limbs, Babinski's sign present on left side, knee and ankle jerks exaggerated on both sides, gait-spastic, ataxic; speech-spastic dysarthria. Blood Kahn negative, discs pale with blurred margins.

Case No. 5. Kalimuthu, Male, 35 years, presenting symptoms were: weakness of left arm and leg with tremors, and involvement of speech. Duration 4 months. Onset was gradual left leg being involved first and a month later, the left arm. Clinical examination revealed the following: weakness of left limbs, increased tone in all four limbs, intention tremors in left limb, knee and ankle jerks exaggerated on both sides, supinator and biceps jerks brisk on both sides, plantar extensor on left side, loss of abdominal reflexes in all four quadrants, epigastric lost on both sides, horizontal nystagmus present, speech - dysarthric. Investigations: negative serology, discs pale.

Case No. 6. Guruswamy, Male, 23 years. Inability to walk with bladder disturbance, of 6 months duration. History of Gonorrhoea 3 years back. On examination, he was found to have weakness of both lower limbs, spasticity of both legs, inco-ordination present in all four limbs, loss of light touch below wrists and ankles with blunting of pain sensation below left knee was noticed, knee jerk absent on right side and ankle jerk lost on both sides, gait - broad based. Investigations showed negative Blood Kahn, C. S. F.—Gl +, Pr. 50, S. 67. 7, Cl. 740.

Case No. 7. Natarajan, Male, 37 years, complained of gradual onset of numbness and tingling over one half of body and face, for a period of 1 year. Clinical examination revealed; diminished power in right limbs increased tone in right arm and both lower limbs, coarse nystagmus present in both eyes, loss of light touch - pain - and vibration sense over right trunk below clavical and upto right costal margin, plantars flexor, deep reflexes in right limbs exaggerated, spurious ankle clonus present. Blood Kahn negative, C.S.F.—Gl.+, Pr. 50, S. too low, Cl. 740.

Case No. 8. Natarajan, Male, 50 years, chief complaint: inability to use all four limbs with pulling sensation all over the body. Duration 1½ years, onset gradual, venereal oistory present. Clinically, speech was stacato, emotional disturbance present, power diminished in all four limbs, tone increased, inco-ordination present in upper and lower limbs, involuntary movements were noticed in the fingers toes, sensations of light touch and pain lost below both clavicles with loss of joint sense. Babinski's sign present on right side, cremasteric lost on left side, tendon jerks in both upper extremities brisk, knee and ankle jerks exaggerated, Rhombergism present, gait - spastic, ataxic; bladder incontinent. Blood Kahn negative, fundus normal.

Case No. 9. Periaswamy, Male, 28 years. Complaint: Difficulty in walking with blurred vision, duration 6 months. Onset gradual, started in

right lower limb with numbness, later on the left lower limb was also involved, history of venereal disease present. Positive findings include: muscle power markedly diminished in both lower limbs and to a certain extent in the right upper, slight inco-ordination noticed in all four limbs, deep reflexes in right upper extremity brisk and in both lower extremities exaggerated, spurious ankle clonus present on right side, bilateral Babinski response, gait ataxic. Investigations showed normal fundus, blood and CSF Kahn negative; L. P. revealed normal pressure, clear fluid, 4 cells per cmm., gl. negative, Pr. 10, S. 70, Cl. 730; Hb. 80%, Serum Proteins - Total 5·75 G% (Alb 2·15, Glob 3·6).

Case No. 10. Pottaiammal, Female, 20 years. Complaint: Parasthesiae in the upper and lower limbs with inability to walk, onset insidious duration 6 months. On examination, the patient had defective hearing. Tone increased in left lower limb, co-ordination impaired in lower limbs, sensations affected: touch lost in both lower limbs, pain lost over same limbs, vibration lost, lower abdominals diminished; deep reflexes in left upper limb diminished and in the lower lost. Plantars flexor. Investigations showed negative Blood and CSF Kahn, 6 cells per cmm., Gl.+, Pr. 90, S. 50, Cl. 700; Fundus - bilateral temporal pallor.

Summary:

1. The ætiology, pathology, symptoms and signs of Disseminated Sclerosis are discussed elaborately.

2. Certain clinical types are also dealt with.

3. A brief mention of the course and prognosis of the condition is made.

4. The diagnosis, differential diagnosis and treatment are reviewed.

5. A record of 10 cases of Disseminated Sclerosis is given.

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CASE NOTES*

A Case of poliomyelitis (Unusual type)

Name: K Age: 2 years.

Sex: Female Admission: 7–3–57

The child had fever for one week before admission. Fever for three days. Later, the child threw one fit confined to the right side. Subsequently, the child was unable to use her right arm and leg with ptosis of right eyelid and slight assymmetry of the face. Deglutition and phonation were also affected.

There was intense pain in all the muscles of the body just after the febrile episode.

Child, on examination was well nourished. The right side of the body was limp on the bed, the left side was reacting near normal.

C. N. S. Ptosis of right eye. III nerve.

Most of the cranial nerves of right side were affected with flaccidity and slight wasting in the muscles.

Spinal:—Lower motor neurone type of paralysis of the right side–Limbs flaccid–No loss of sensation–Right side weekness of limbs but not complete.

Pupils: Normal–equal–reactive.

Abdominals : ?

Plantar: Flexor.

Neck mucles slightly rigid : ? Pain.

Other reflexes: Absent right side.
Left side: Sluggish.

C.S.F. Proteins: 60 mg%
Fluid slightly under pressure.

Clobulin: +
No cobweb formation.

Chlorides: 740%
Cell count: 4/CMM.

Sugar: Low
No A F B seen in smear.

Blood: No leucocytosis.
Otherwise normal.

Urine: Normal Fundus: Normal

The Child was put on expectant treatment with a broad spectrum antibiotic cover to avoid intercurrent infection, nourished by nasal tube and by Intravenous medications. There was slow recovery, the left side recovered completely with full power but the right side of the body retained its weakness though much less than at the onset.

Contractures were prevented by passive movements and splinting. Swallowing and phonation returned slowly.

The case was shown here to bring out the peculiar nature of the attack

(a) resembling hemiplegia by affecting one side of the body, but of the lower motor neurone type.

(b) the simultaneous affection of the **spinal** and **medullary** **necleii** and ev en that on one side.

(c) the complete recovery of the affected side (left).

(d) the commencement of the attack with fits which is common with encephalomyelitis but where the neurological defect is motor.

Discussion:—Acute anterior poliomyelitis is a virus disease and attacks the motor horn cells of the spinal cord and the brain stem.

* Reprinted from the Proceedings of the North Arcot District Clinical Society 1957.

Infection is by the gastro-intestinal tract. The anterior horn cells undergo rapid chromatolyis and the muscles that they supply waste. But the destruction is patchy and ultimate destruction of cells is found even though at the onset a grim picture is presented. Recovery of most of the cells is the rule. There is no loss of sensation as the posterior horn cells of the tracts are untouched.

There is a general febrile toxaemia with fever ranging from 102 to 104. There may be signs of MENINGEAL irritation and MUSCLES are TENDER. Symptoms subside in 24 to 48 hours and in many cases complete recovery and no paralysis is the rule. This is the non-paralytic form.

There may or may not be a second hump of fever and it may pass on to paralytic stage. But the damage to be done has already been done in the first bout of fever itself. Temperature comes down but the muscle pains and tenderness continue and paralysis sets in. A flaccid type of paralysis asymmetrical occurs and wasting follows soon.

In one week, most of the muscles recover their power and tone and improvement can continue for some months and the residuary defect will be known only then.

A muscle showing no activity after one month is not likely to recover. The cerebro spinal fluid changes are as described in the case. The dangers are from the attack of the muscles of deglutition and respiration (bulbar form). In the bulbar form, swallowing is affected with aspiration pnumonias. Respiration gets affected with cyanosis with the necessity for artificial respiration in an iron lung.

Later, the stage of contracture comes on with unequal recovery of opposing muscles with resultant deformities and contractures. There is no sensory loss but loss of vosomotor instability occurs as coldness of limbs, or flushing, indiscriminate sweating, cyanosis of the extremities or even arrest of growth of bone.

Diagnosis:—From the number of cases that generally occur together. The meningeal irritation at the outset may suggest meningitis, the muscle pains rheumatism, the motor paresis suggest infective polyneuritis.

Progress is good in most cases except the bulbar forms and in the other forms most of the muscles recover and the ultimate deformity and paralysis is limited.

Prophylaxis is done with salk type of vaccines as well as temporary immunity with gamma globulins. Tonsillectomies and other injections are to be avoided during the seasonal prevalence.

Treatment:—Rest in bed, sedatives. Complete rest during the febrile state limits the paralysis remarkably.

The position of the limbs should be in optimium to avoid contractures. Passive movements to counteract spasm. Watch out for respiratory difficulties. In bulbar paralysis the patient should be kept in a prone position to avoid sucking in of saliva and other secretions into the lung. Contractures developing later are to be treated by orthopaedic procedures and loose flail joints can be corrected by caliper shoes or even by arthodesis in selected cases.

A case of Rhinoplasty

Plastic surgery aims at the correction or reconstruction of parts deformed or lost by injury, disease or congenital deformity and reconstructive rhinoplasty is an important branch of this aspect of surgery. In the following case, a nose deformed by injury was brought to its original shape by operative reconstruction carried out in many stages.

Periapayyan, male, aged 25 years undergoing a term of imprisonment in

the Central Jail, Vellore, had his nose cut off by a fellow convict in the month of March 1956. Attempts at primary repair suturing the cut portion to its original attachment having failed, he was referred to this hospital for reconstructive surgery. He was admitted here in July 1956 and it was decided to raise a pedicle graft from the abdominal wall and transfer it to the nose through the medium of the forearm. Accordingly as the first stage, a tubed pedicle graft was raised on the right side of the abdomen. In September 1956, the outer end of the tubed flap was separated from its attachment and applied to and sutured in position on the radial aspect of the right forearm above the wrist. On 19th January, 1957, under endotracheal anaesthesia, the graft was transferred to the nasal area. This was accomplished as follows :—

The abdominal attachment of the pedicle graft was freed and the arm was abducted so that the free end of the graft could be applied to the nasal region. The edges of the nasal scar were freshened by careful excision and the free end of the pedicle graft was so cut and shaped that on suturing it to the nasal area, the walls and septum of the nose with the external nares could be formed. Two small rubber tubes were inserted into the external nasal openings and the abducted arm was immobilised in position by P. O. P. On 18—2—1957 the arm was released and the reconstruction of the nose completed by trimming and suturing the upper portion of the graft. At the time of presentation of the case, the shape of the nose was normal, though the colour of the grafted area was slightly darker. The patient was able to breathe and smell properly.

Discussion:—This is an example of plastic surgery where a flap from a distance (viz. the abdominal wall) is transferred through an intermediate host (viz. the forearm) to the area to be repaired.

In plastic surgery, reconstruction is usually accomplished by employing free grafts or skin flaps. The commonest use of free grafts is in skin-grafting where a split-skin-graft (Thiersch) or a full thickness graft (Wolfe) may be employed. Other types of free grafts are the fat graft fascia lata graft, cartilage graft, chondrocutaneous graft and the bone graft. Repair by means of flaps is very useful and elegant and in general there are two types of flaps, the local flap which is a single stage surgical procedure, and the flap from a distance which may require two or more stages for its completion. The local flap makes use of tissue adjacent to the defect to be closed and it is transferred, after thorough mobilisation, by advancement, rotation or transposition. If the secondary defect left after transposition of the flap can not be closed without undue tension, a suitable free graft has to be applied. Flaps from a distance utilise tissue not immediately adjacent to the defect to be closed. They may be transferred directly without an intermediate host such as from the forehead to the nose or cheek, or from the neck to the face. In many cases, with the forearm or wrist as the carrier, flaps may be transported to any part of the body surface and large areas of tissue may be moved by this method. In the case under discussion, the latter method was used.

In transferring a flap from one part of the body to another, it is necessary to pay particular attention to accurate design and to preserve its blood supply. The quantity, character and shape of lost tissues are all taken into consideration in planning the type of repair During the operation. the necessity for gentle atraumatic handling of tissues, perfect haemostasis. fine sutures and prevention of infection should be remembered.

The three chief aims in plastic surgery are :—

(i) Restoration of shape.

(ii) Restoration of function ; and

(iii) Attainment of a good cosmetic result.

In the case of reconstructive rhinoplasty discussed here, all these aims have been successfully achieved.

Resolutions passed at the Open Session of the 13th Madras State Medical Conference on 1-12-1958.

1. In view of the rapid industrialisation of our State and of the Occupational Health Problems that will inevitably arise, this Conference requests the State Government to create a separate department for Occupational Health Services and also to create a Directorate for Occupational Health Services.

Moved by : Dr. A. G. Leelakrishnan, Coimbatore.
Seconded by : Dr. N. R. Subrahmanyam, Tanjore.

2. In view of the various handicaps suffered by the Medical Licentiates and in the interests of higher medical education, this Conference requests the State Government and the University of Madras to reopen the condensed MBBS Course for Licentiates in modern medicine.

Moved by : Dr. V. Raghavendra Rao, Salem.
Seconded by : Dr. T. Narayanaswami, Salem.

3. This Conference requests the State Government to convert the presen College of Integrated Medicine to a College of Modern Medicine and to institute in it Post Graduate Courses and Research Studies in the different branches of the Indian Medicine.

Moved by : Dr. A. G. Leelakrishnan, Coimbatore.
Seconded by : Dr. G. Ramachandramurthi, Madras.

4. This Conference resolves to request the Government to include all registered medical practitioners as belonging to "Essential Services" for the purpose of Rent Control Act.

Moved by : Dr. V. Vijayaraghavan, Madras.
Seconded by : Dr. V. S. Selvapathi, Madras.

5. This Conference resolves to request the Government to increase the scale of pay of Service Men as recommended by the Central I. M. A.

Moved by : Dr. V. Vijayaraghavan, Madras.
Seconded by : Dr. C. Rama Rao, Madras.

6. This Conference resolves that the "Husband and Wife" Doctor Members in service as far as possible be kept in the same station when question of transfers are considered.

Moved by : Dr. V. Vijayaraghavan, Madras.
Seconded by : Dr. B. Rama Rau, Madras.

7. This Conference resolves to request the Government to abolish the present system of "authorised medical attendants" and allow the Government Servants to choose their own medical attendants for purposes of reimbursement, leave etc.,

Moved by : Dr. V. Vijayaraghavan, Madras.
Seconded by : Dr. A. S Vaidyanathan, Madras.

8. It is resolved to request the Editor of the Journal of the Indian Medical Association, Calcutta, to have a list of priority for the articles and to record the date of receipt of the article when it is published.

Moved by : Dr. B. Ramamurthi, Madras.
Seconded by : Dr. G. Ramachandramurthi, Madras.

9. It is resolved to request the Editorial Committee of the Journal of the Indian Medical Association to drop the advertisement appearing on the front cover page of the Journal of the Indian Medical Association.

Moved by : Dr. B. Ramamurthi, Madras.
Seconded by : Dr. B. Rama Rau, Madras.

10. This Conference requests the State Government to expedite the elections to the Indian Medical Councils otherwise the State and the Profession will lose its representation after the 1st April 1959.

Moved by : Dr. A. G. Leelakrishnan, Coimbatore.
Seconded by : Dr. S. R. Rajaram, Salem.

11. This Conference requests the Corporation of Madras to name the new Over Bridge in Chetpet after the late Dr. M. R. Guruswami Mudaliar.

Moved by : Dr. M. Santosham, Madras.
Seconded by : Dr. P. Alagasingari Naidu, Madras.

12. This Conference expresses its grateful thanks to the several organisations, officers, firms and the authorities of the Madras Medical College for their very kind help and co-operation with the organisers of this Conference and making the Conference a grand success.

Moved by : Dr. S. R. Rajaram, Salem.
Seconded by : Dr. A. G. Leelakrishnan, Coimbatore.

C. ARUMUGAM,
Honorary Secretary,
INDIAN MEDICAL ASSOCIATION,
MADRAS STATE BRANCH.